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Etiology of the Austin Flint Murmur

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Objectives. **The aim of the study was to determine the mechanism of the Austin Flint murmur.**

Background. **More than 100 years after the initial description of the Austin Flint murmur, the etiology of the murmur remains unclear.**

Methods. **M-mode and two-dimensional echocardiography, conventional and color flow Doppler study, and cine nuclear magnetic resonance (cine NMR) imaging were performed in 24 patients with clinically moderate or severe aortic regurgitation. Mitral valve area was determined by planimetry and pressure half-time measurement. Overlap of the aortic regurgitation and mitral inflow jets was graded 0 (no overlap) to 4 (marked overlap) by Doppler study and cine NMR imaging. The volume of signal loss resulting from turbulent blood flow secondary to the aortic regurgitation jet was determined on cine NMR images, and the extent of contact with the left ventricular endocardium was graded 0 (no contact) to 4 (extensive contact).**

Results. **The presence of an Austin Flint murmur did not correlate with mitral valve area (2.7 ± 0.8 cm^2 with the murmur vs. 2.5 ± 0.7 cm^2 without), overlap of the aortic regurgitation and mitral inflow jets (3 ± 1 vs. 2.3 ± 1.2), diastolic mitral regurgitation (50% vs. 71%) or fluttering of the anterior mitral valve leaflet (70% vs. 50%). The presence of an Austin Flint murmur correlated best with the volume of signal loss associated with the aortic regurgitation jet on cine NMR imaging (65 ± 16 ml with the murmur vs. 38 ± 11 ml without, $p < 0.001$) and the extent of contact of this signal loss with the left ventricular endocardium (2.9 ± 0.5 vs. 1.5 ± 0.4, $p < 0.0001$).**

Conclusions. **The Austin Flint murmur is caused by the aortic regurgitation jet abutting the left ventricular endocardium, resulting in the generation of a low-pitched diastolic rumbling.**

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In 1862, Austin Flint described a "presystolic blubbering" in two patients with severe aortic regurgitation and a normal mitral valve (1). In the years that followed, apical diastolic rumbling murmurs in patients with aortic regurgitation were noted to be presystolic, mid-diastolic or holodiastolic and were given the eponym of Austin Flint murmurs.

Austin Flint, most frequently remembered for the murmur that bears his name, was ironically, a critic of associating physical signs with the name of the original describer. He stated that "so long as signs are determined from fancied analogies, and named from these or after the person who describes them, there cannot but be obscurity and confusion" (2). These words were prophetic, because >100 years after the original description of the Austin Flint murmur its etiology remains unclear. Proposed etiologies have included functional mitral stenosis (1,3), increased turbulence within the left ventricle secondary to mixing of mitral inflow and aortic regurgitation (4,5), diastolic mitral regurgitation (6,7), fluttering of the anterior mitral valve leaflet (8,9) and low pitched vibrations of the aortic regurgitant murmur best heard at the apex (10).

Because recent innovations in cardiac imaging have made it possible to noninvasively examine these possibilities, we studied 24 patients with aortic regurgitation in an attempt to elucidate the etiology of the Austin Flint murmur.

Methods

Study patients. Patients with clinically moderate or severe aortic regurgitation were recruited from the University of California, San Francisco and the San Francisco General Hospital cardiology services. Twenty-four patients with a mean age of 45 ± 16 years were studied; 20 were male and 4 female. Informed consent was obtained using a protocol approved by the Committee on Human Research. All patients had at least clinically moderate aortic regurgitation as assessed by duration and intensity of their murmur, increased cardiac size and pulse pressure >65 mm Hg. Nineteen of the 24 patients had a previous radionuclide scan, all showing a regurgitant fraction ≥50%.

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Clinical examination. Cardiac examination and determination of the presence of an Austin Flint murmur were performed by an experienced auscultator (M.D.C.). The murmur was documented before the echocardiographic and nuclear magnetic resonance (NMR) studies were performed.

Echocardiography. Two-dimensional echocardiographic and Doppler evaluations were performed on an Irex Meridian using 2- and 3.5-MHz transducers. Patients were imaged from the parasternal and apical windows in the 90° left lateral decubitus position. An M-mode tracing of the mitral valve was obtained from the parasternal window, with a scroll speed of 75 mm/s. Mitral and aortic flow were examined, with angular and translational adjustments of the transducer made aimed at placing the beam axial to flow while optimizing velocity and definition of the Doppler signals. Pulsed Doppler mapping of the penetration and direction of the aortic regurgitant jet was performed in the parasternal and apical views. Pulsed Doppler mapping of the mitral diastolic inflow signal was performed with the sample volume positioned immediately downstream from the mitral valve leaflets, for pressure half-time analysis. Continuous wave Doppler echocardiography was used to obtain the aortic regurgitant envelope for measurement of pressure half-time. Color Doppler examinations were performed on an Aloka 880 with 2.5-MHz transducers. Adjustments were made to maximize the aortic regurgitant jet recorded in the parasternal and apical views while minimizing background artifact.

Cine NMR imaging. Cine NMR images were acquired on a GE Signa unit with 1.5-tesla field strength using methods described previously (11,12). Utilizing shallow flip angles of 30°, short echo delay times of 12 ms and pulse repetition times of 21 ms, 14 to 20 images were obtained per cardiac cycle. Images of 10-mm slice thickness were acquired repeatedly in the axial plane at three levels simultaneously until the entire heart was encompassed from apex to pulmonary artery bifurcation. Two patients were unable to undergo NMR imaging because of routine exclusionary criteria (claustrophobia and a history of possible ophthalmic metal splinters from occupational welding).

Data analysis. All measurements (echocardiographic and cine NMR) were performed off-line by observers who had no knowledge of the presence or absence of an Austin Flint murmur. Mitral valve area was determined by the Doppler pressure half-time method and also by planimetry of the mitral valve in the echocardiographic parasternal short-axis view at early diastole (peak opening) and end-diastole (one frame before the R wave) at the level of the valve leaflet edges.

Overlap of the mitral inflow and aortic regurgitant jets was determined by pulsed and color Doppler study as well as by cine NMR imaging. Mixing of the Doppler jets was graded subjectively on a scale of 0 to 4 (0 = none, 1 = mild, 2 = moderate, 3 = moderate to marked and 4 = marked). Cine NMR assessment of directionality of the aortic regurgitant jet in relation to the mitral valve orifice was similarly graded on a 0 to 4 scale (0 = no overlap of the mitral valve orifice, 4 = complete overlap of the mitral valve orifice). Cine NMR images were reviewed on two separate occasions without knowledge of prior results. Exact agreement regarding the directionality of the aortic regurgitant jet was present in 16 of 23 cases. There was no disagreement >1 grading point. On the second reading, the interpretation was 1 grading point higher in four cases and 1 grading point lower in three cases.

Diastolic mitral regurgitation was evaluated by Doppler and color Doppler interrogation inside the mitral valve in the mitral anulus. Fluttering of the anterior mitral valve leaflet was determined from the hard copy M-mode echocardiographic recordings of the mitral valve. Echocardiographic severity of aortic regurgitation was graded on a scale of 1 (mild) to 4 (severe), as determined by the area of the aortic regurgitation jet in multiple planes by both pulsed and color Doppler study, pressure half-time of the aortic regurgitation jet and the size of the regurgitant jet orifice.

The cine NMR images were displayed on a computer monitor in cinematic fashion to evaluate cardiac function and changes in blood pool signal intensity during the cardiac cycle. The shape, extension and timing of areas of altered signal intensity were noted. The diastolic image showing the most extensive and intense signal loss was visually identified and photographed. The area of signal loss was outlined by using a track ball cursor yielding the cross-sectional area at this level. This procedure was repeated on images at the same point in the cardiac cycle at each anatomic level in which signal void was visible in order to obtain the maximal volume of diastolic signal loss for each patient. The extent of aortic regurgitant signal loss abutting the left ventricular endocardium was subjectively assessed by three observers independently and graded on a 0 (no contact) to 4 (extensive contact) scale (Fig. 1).

Statistical analysis. Data are presented as mean value ± 1 SD. Patients with and without an Austin Flint murmur were compared by using two-tailed unpaired *t* testing. Linear regression analysis was performed to ascertain those factors most closely related to the presence of the Austin Flint murmur. A p value <0.05 was considered significant.

Results

The Austin Flint murmur was present in 10 of 24 patients studied. The murmur was mid-diastolic in 9 of 10 patients and presystolic in all 10 patients. The presence of an Austin Flint murmur correlated with a younger age ($p = 0.05$) and the presence of a third heart sound on examination ($p < 0.01$). The Austin Flint murmur tended to correlate with echocardiographic severity ($p = 0.09$), but not with pulse pressure (Table 1).

Mitral valve area. Mitral valve area, as determined by the pressure half-time formula, was decreased in the group as a whole. However, it was not significantly (p = NS) different in patients with compared with those without an Austin Flint murmur (2.7 ± 0.8 cm^2 in those with a murmur vs. 2.5 ±

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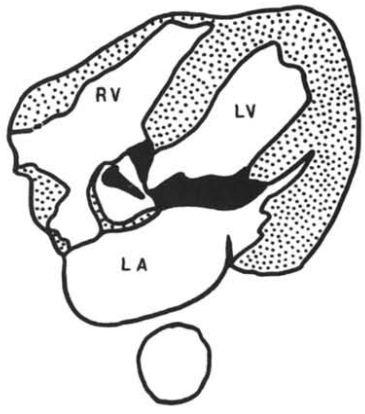
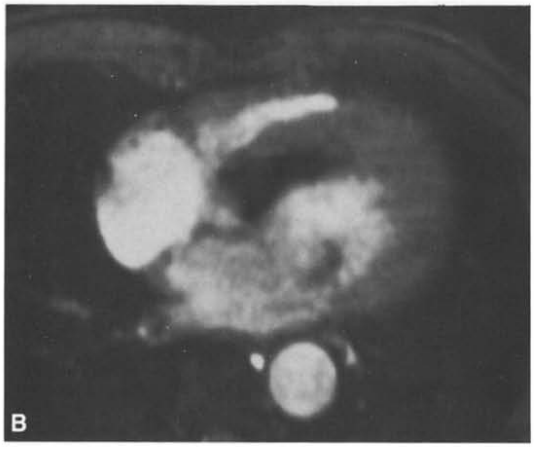
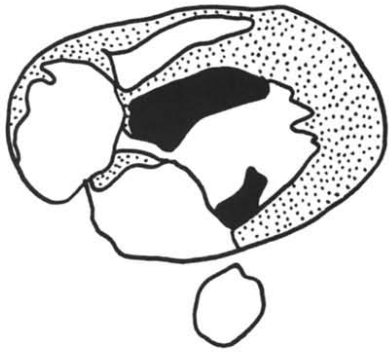
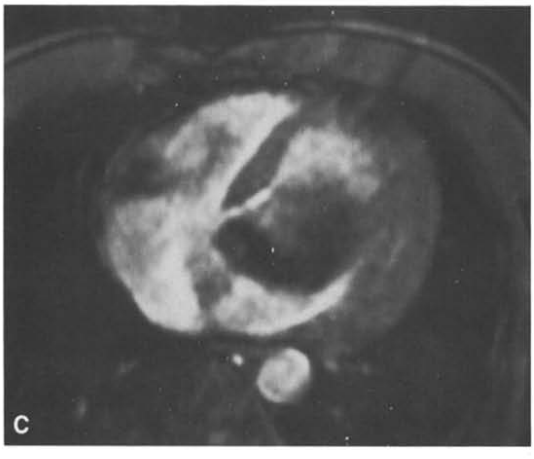








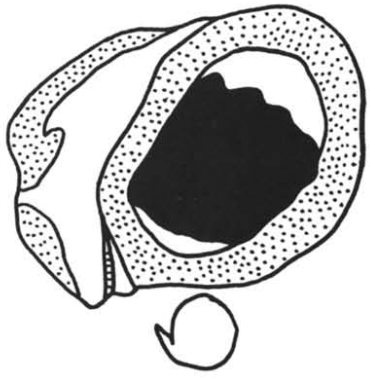


Figure 1. Cine nuclear magnetic resonance (NMR) images, obtained in the transaxial plane, at the mid ventricular level. **A** and **B**, From patients with a mild to moderate jet of aortic regurgitation signal loss. In **A**, there is minimal contact of the regurgitant jet with the endocardial surface; the Austin Flint murmur was absent. In **B**, there is extensive contact of the regurgitant jet with the septal myocardium; the Austin Flint murmur was present. **C** and **D**, From patients with a moderate to severe jet of aortic regurgitation signal loss. In **C**, although an extensive area of signal loss is noted, contact with the left ventricular endocardium is minimal; the Austin Flint murmur was absent. In **D**, extensive contact with septal and lateral wall left ventricular myocardium is noted; the Austin Flint murmur was present. LA = left atrium; LV = left ventricle; RV = right ventricle.

Table 1. Correlations With the Presence or Absence of an Austin Flint Murmur

	Austin Flint Murmur		
	Present	Absent	p Value
Age (yr)	41 ± 12	52 ± 13	0.05
Pulse pressure (mm Hg)	80 ± 22	81 ± 16	NS
Third heart sound	60%	7%	<0.01
Severity of AR on echocardiography (1 to 4)	3.0 ± 0.7	2.1 ± 1.4	0.09
Mitral valve area (cm^2)	2.7 ± 0.8	2.5 ± 0.7	NS
Overlap of AR and mitral inflow (0 to 4)	3.0 ± 1.0	2.3 ± 1.2	NS
Diastolic mitral regurgitation (% of patients)	50	71	NS
Fluttering anterior mitral leaflet (% of patients)	70	50	NS
Signal loss (cine NMR imaging) abutting LV endocardium (0 to 4)	2.9 ± 0.5	1.5 ± 0.4	<0.0001

AR = aortic regurgitation; LV = left ventricular; NMR = nuclear magnetic resonance.

0.7 cm^2 in those without). Mitral valve orifice area by planimetry was normal at the onset of diastole in both groups (4.8 ± 1.4 cm^2 in those with a murmur vs. 5.2 ± 1.1 cm^2 in those without, p = NS); however, by end-diastole, mitral valve area was significantly decreased in both groups (2.8 ± 0.6 cm^2 in those with vs. 3.1 ± 0.6 cm^2 in those without, p = NS).

Mixing of mitral inflow and aortic regurgitant jets. There was no significant difference in the overlap of the aortic regurgitation and mitral inflow jets, as graded (0 to 4) by pulsed and color Doppler study, between patients with an Austin Flint murmur (3 ± 1) and those without (2.3 ± 1.2). Confirmation of this finding was obtained by cine NMR grading (0 to 4) of the directionality of the aortic regurgitant jet with relation to the mitral valve orifice. No significant difference existed between patients with (2.0 ± 1.5) versus those without (1.7 ± 1.3) an Austin Flint murmur.

Diastolic mitral regurgitation. The presence of diastolic mitral regurgitation, as assessed by Doppler interrogation for retrograde signals just inside the mitral valve leaflets, was not significantly different between patients with (50%) versus those without (71%) an Austin Flint murmur. Cine NMR diastolic signal loss across the mitral valve did not occur with different frequency in the two groups (40% in those with vs. 33% in those without an Austin Flint murmur).

Fluttering of the anterior mitral valve. The frequency of fluttering of the anterior mitral valve leaflet was not significantly greater in patients with (70%) than in those without (50%) an Austin Flint murmur.

Signal loss characteristics on cine NMR imaging. The volume of signal loss associated with the aortic regurgitant jet in patients with an Austin Flint murmur (65 ± 16 ml) was significantly greater than in those without (38 ± 11 ml, p < 0.001). Three observers independently determined the extent of contact between the aortic regurgitant jet signal loss and the left ventricular endocardium (Fig. 1). For each observer and for the averaged results (Table 2) there was a significantly greater extent of signal loss in contact with left ventricular endocardium in patients with an Austin Flint murmur (2.9 ± 0.5) than in those without (1.5 ± 0.4, p < 0.0001). An area of signal loss of the aortic regurgitant jet in contact with the left ventricular endocardium ≥2.6 allowed discrimination of the presence or absence of the Austin Flint murmur in 21 of the 22 patients (Fig. 2). Using multiple regression analysis, the extent of signal loss in contact with the left ventricular endocardium had the strongest association with the presence of an Austin Flint murmur, with no additional association with the echocardiographic severity of aortic regurgitation, directionality or volume of the aortic regurgitant jet.

Table 2. Interobserver Assessment of Extent of Cine NMR Caused by Abutment of the Aortic Regurgitant Jet Against Left Ventricular Endocardium

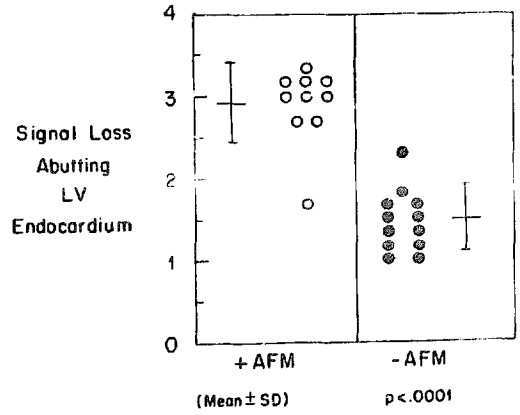
	Observer 1	Observer 2	Observer 3	Mean
Austin Flint murmur				
Present	3.4 ± 0.7	2.6 ± 0.4	2.8 ± 0.6	2.9 ± 0.5
Absent	1.8 ± 0.6*	1.3 ± 0.4*	1.3 ± 0.5*	1.5 ± 0.4*

*p < 0.0001 versus values with an Austin Flint murmur present. All values are expressed as mean values ± SD. Abbreviation as in Table 1.

Discussion

In his paper "On Cardiac Murmurs," (1) Austin Flint described the clinical and postmortem findings in two patients with aortic regurgitant murmurs and presystolic murmurs at the base of the heart: "At one time I supposed this blubbering murmur denoted a particular lesion, viz., adhesion of the mitral curtains at their sides, forming that species of mitral contraction known as the buttonhole slit; but I have found this variety of murmur to occur without that lesion,

Figure 2. Distribution of the extent of contact of the aortic regurgitant jet with the left ventricular (LV) endocardium in patients with (+) and without (−) an Austin Flint murmur (AFM) (averaged results of three observers).



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and, in fact . . . when no mitral lesion whatever exists." Both of his patients had a normal mitral valve at the time of autopsy. Flint postulated that in patients with considerable aortic regurgitation, the mitral leaflets are brought into premature diastolic coaptation, such that when atrial contraction occurred, it would cause the presystolic "blubbering murmur" otherwise seen with mitral stenosis.

Proposed etiologies for the Austin Flint murmur. Fortuin and Craige (3), utilizing simultaneous phonocardiograms, apexcardiograms and mitral valve echocardiograms, found that both the mid-diastolic and presystolic components of the Austin Flint murmur occurred as the mitral valve was closing. We have also found that the mitral valve begins to close prematurely in patients with considerable aortic regurgitation, however, this functional mitral stenosis occurs in patients both with and without an Austin Flint murmur.

Laniado and colleagues (4) evaluated nine open chest dogs with acute aortic regurgitation and a low pitched diastolic ventricular murmur. They found only a slight decrease in mitral valve area in these dogs, and postulated that the major determinant of the turbulence responsible for the Austin Flint murmur was mixing of the mitral inflow with the aortic regurgitant jet. Utilizing pulsed wave and color Doppler studies and cine NMR imaging we did not find a significant difference in the admixture of the mitral inflow and aortic regurgitant jets in patients with and without an Austin Flint murmur.

There have been conflicting reports (5–9) as to the presence of diastolic mitral regurgitation and fluttering of the mitral valve leaflets in patients with an Austin Flint murmur. We did not find a significant difference in the frequency of diastolic mitral regurgitation or fluttering of the mitral valve leaflets in patients with and without such a murmur.

It has been suggested (10) that the Austin Flint murmur might be due to apical echoing of the aortic diastolic murmur. Using cine NMR imaging, we found that the extent of signal loss of the aortic regurgitant jet in contact with the left ventricular endocardium was strongly associated with the presence of an Austin Flint murmur. The extent of contact correctly predicted the presence or absence of the murmur in all but one patient.

The mechanism of cardiac murmurs is not fully understood. Postulated etiologies have included turbulence, eddy formation and vortex shedding around an obstruction. The most common mechanism is thought to involve turbulent disturbed blood flow, which can be generated by a high velocity jet passing through a narrow orifice, by two streams intersecting or by a jet striking a surface (13). Signal loss on cine NMR imaging has been shown to be proportional to the degree of turbulence. Using models of laminar and turbulent flow, Evans et al. (14,15) found that as the intensity of turbulent flow increased, there was a threshold above which signal intensity decreased linearly as turbulence increased. These observations occurred at physiologic velocities and Reynolds numbers. This phenomenon is thought to occur as a result of "eddy diffusion" with areas of turbulence leading to dephasing of spins and therefore signal loss in the magnetic resonance image.

In this study, we observed that the extent of signal loss of the aortic regurgitant jet in contact with the left ventricular endocardium was strongly predictive of the presence or absence of an Austin Flint murmur. We hypothesize that when the aortic regurgitant jet strikes the trabeculated left ventricular endocardium, the turbulence in the regurgitant jet increases. The murmur is caused either by transmission of the aortic regurgitant turbulence or by vibration of the left ventricular wall. Auscultation of the murmur would therefore depend on the degree of turbulence (signal loss) and proximity of this turbulence to the left ventricular wall in addition to the physical habitus and acoustic properties of the patient.

Limitations of the study. Determination of the presence of an Austin Flint murmur is subjective, belonging to the realm of the art of physical examination. Unfortunately, there is no reference standard for its presence, and one must rely on the experience of recognized experts.

The use of the pressure half-time to assess mitral valve area in the setting of moderate to severe aortic regurgitation has been supported by some studies; others (16,17) have stated that it overestimates mitral valve area. Nonetheless, any overestimation of valve area should have been equal in the two groups. Mitral valve area was also calculated by planimetry, which is believed to be accurate even in the setting of aortic regurgitation; the resulting values did not differ between patients with and without an Austin Flint murmur either at the onset of diastole at its maximal opening or at the end of diastole.

Conclusions. We conclude that the Austin Flint murmur is not caused by functional mitral stenosis, overlap of the aortic regurgitation and mitral inflow jets, diastolic mitral regurgitation or fluttering of the mitral valve leaflets. The presence of the Austin Flint murmur strongly correlates with the extent of the aortic regurgitant diastolic signal loss on cine MR images in contact with left ventricular endocardium. We hypothesize that the Austin Flint murmur is caused by abutment of the aortic regurgitant jet against the left ventricular endocardium, which results in the generation of a low pitched diastolic rumbling murmur.

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